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Epidemiology of Testicular Cancer in Oklahoma and the United States

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Abstract

Testicular cancer is a rare cause of morbidity and mortality in the US. Marked disparities in the development of this cancer exist, with testicular cancer being more common in Caucasian men and men of higher socioeconomic status. The incidence of testicular cancer is increasing worldwide, and the reasons for this have not been well documented. It has been proposed that this increase may be due to highly prevalent environmental factors, or from exposure to polychlorinated biphenyls, polyvinyl chloride, cigarette smoking, and tetrahydrocannabinol (THC). For our analysis, data were obtained from the Oklahoma Central Cancer Registry and the Surveillance, Epidemiology and End Results program. Age-adjusted incidence rates and five-year relative survival were calculated for Oklahoma and for the US. Overall, incidence was lower in Oklahoma than the US, but no differences were observed between the US and Oklahoma regarding survival by year of diagnosis, race, age, and stage.

BACKGROUND

Malignant tumors of the testis are rare, with testicular cancer being the 25th most common cause of cancer in the US and comprising 0.5% of all new cancer cases.[1] Testicular cancer is a disease of young men compared to many other cancers, with a median age at diagnosis of 33.[1] A male in the US has a 0.4% chance of being diagnosed with testicular cancer in his lifetime.[1] In 2012, it was estimated that there were 233,602 men living with testicular cancer in the US[1] Of the 8,430 new cases of testicular cancer expected in 2015, only 380 deaths are expected.[1] Testicular cancer has a five-year relative survival rate of 95.3%. However, the highest percent of deaths occurs among men aged 20-34, and the median age at death is 41.[1]

The testes function as part of the male reproductive system, participating in spermatogenesis and in the production of androgens, primarily testosterone.[2] The primary cell types in the testis are the seminiferous tubules, where germ cells participate in spermatogenesis, and the interstitial, or Leydig, cells which produce testosterone. Of all primary tumors of the testis, 90-95% are germ cell tumors. Germ cell tumors are either seminomas or nonseminomas. Seminomas tend to be slow-growing tumors that are typically confined to the testes and are typically identified in men between 20 and 30 years of age. The nonseminomatous germ cell tumors are choriocarcinomas, yolk sac tumors, and teratomas, which tend to grow more

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quickly and be more aggressive compared to seminomas. The remaining 5-10% of testicular tumors are nongerminal neoplasms, or stromal tumors, and consist of Leydig cell tumors, Sertoli cell tumors, and gonadoblastomas.[2] Stromal tumors are rare, are typically not cancerous, and are usually found in children.[3]

There is marked variation in the development of testicular cancer among different countries, races, and socioeconomic classes. In the US, the 2012 incidence rate for testicular cancer was 6.7 per 100,000 white men, 1.5 per 100,000 black men, and 4.9 per 100,000 for Hispanic men.[1] Notable differences in the incidence of testicular cancer have also been observed in Europe, both between and within countries. For example, there was a stark difference in incidence between neighboring countries Finland (2.5/100,000) and Denmark (9.2/100,000), as well as large differences between regions of the same country, with rates ranging from 2.8 to 7.9 in different parts of France.[4] Many studies have shown that high socioeconomic status is associated with a higher risk of testicular cancer.[5,6,7]

Furthermore, the incidence of testicular cancer is increasing worldwide.[4,8] The US has seen a significant Annual Percent Change (APC) in the rates of testicular cancer between 1973-2007.[9] From 1973-1977 the incidence rate for Caucasian men was 4.0 per 100,000, which increased to 6.4 between 2003-2007, for an APC of +1.4%. The US incidence rate for black men was 0.8 per 100,000 men between 1973-1977, which increased to 1.1 per 100,000 men in 2003-2007, for an APC of +1.9%.9 According to the SEER statistics, the age-adjusted incidence rate for US men (all races) was 5.6 per 100,000 men between 2010-2012.[1] The death rate from testicular cancer for the same period was 0.3 per 100,000 men.[1]

The reason for this increased incidence in testicular cancer is unclear. Improved diagnostic techniques and known risk factors, such as cryptorchidism, HIV infection, and fetal estrogen exposure are unlikely to explain this trend. Some have suggested that this increase in incidence may be due to the presence of highly prevalent environmental factors, which may act as endocrine disrupters.[10] These distort the normal hormonal activity of the testis and altering cellular development.[8] The organochlorine p,p'-DDE is a pesticide that was commonly used until it was banned in the 1980's. P,p'-DDE is an androgen receptor antagonist, and in a study by Guo et al. (2005), exposure to pesticides was linked to a higher incidence of testicular cancer.[10] A case-control study by McGlynn et al (2008) also associated higher plasma levels of both p,p'-DDE and chlordane with the development of testicular germ cell tumor.[11] Other substances that have been proposed to be associated with an increased risk of testicular cancer are the polychlorinated biphenyls,[12] polyvinyl chloride,[13] and cigarette smoking.[14]

Of all of the substances postulated to be associated with the development of testicular cancer, the one which has been most clearly demonstrated is tetrahydrocannabinol (THC).[14] Several studies have demonstrated an association between having ever smoked marijuana,[15] early (<18 years of age), daily, frequent (>1 time per day) and long-term use of marijuana.[16] The mechanism for this increased risk has not yet been well-described, but cannabinoid receptors are found in the hypothalamic-pituitary-gonadal axis, and are found on both Leydig and germ cells in the testicle.[17] This is an interesting problem facing epidemiologists

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and urologists today, and certainly more research into the causes and prevention of this disease are necessary.

Although variation in the development of testicular cancer has been described in the US and worldwide, the descriptive epidemiology of testicular cancer has not been described in Oklahoma. The aim of this analysis was to provide a description of the incidence and survival of testicular cancer, in the US and Oklahoma.

METHODS

We obtained data on testicular cancer incidence from the Oklahoma Central Cancer Registry (OCCR), which included all cases of cancer diagnosed in Oklahoma residents from 1997-2012. The OCCR is a participant in the National Program of Cancer Registries and follows all guidelines of the North American Association of Central Cancer Registries. Data for the US were obtained from the Surveillance, Epidemiology, and End Results (SEER) program during the same time period, which was designed to be representative of the US population.[18] We restricted the cases and population to males for calculation of incidence rates. We used SEER site recodes to restrict our analysis to men with testicular cancer, which included International Classification of Diseases of Oncology, Third Edition/World Health Organization (ICDO-3/WHO) 2008 site codes of C620-C629, excluding histology codes of 9050-9055, 9140, 9590-9992 (lymphomas, Kaposi sarcoma, and mesothelioma).[19] We obtained the variables of race, ethnicity, year of diagnosis, and age from the OCCR and SEER registries for Oklahoma and the US, respectively. We classified race/ethnicity as white non-Hispanic (NH), African American NH, American Indian/Alaska Native NH, Asian/Pacific Islander NH, and Hispanic. For Oklahoma, the OCCR data were linked with Indian Health Service records to better classify American Indians/Alaska Natives. We used the Derived SEER Summary Stage 2000 variable to classify stage as localized, regional, and distant stage in Oklahoma from 1998-2012 and Summary Stage 2000 for stage using SEER data. In our analysis of stage at diagnosis, we excluded those reported by a death certificate only or on autopsy.

To calculate age-adjusted incidence rates (AAIR), we used the 2000 Standard Population. We calculated AAIR by year of diagnosis, grouped into 1997-2001, 2002-2006, and 2007-2012 for stability purposes. We also calculated AAIR by race/ethnicity using the categories specified above. Additionally, we calculated age-specific incidence rates, classifying those 0-14 years of age at diagnosis in one group, separating those who were 15-64 at diagnosis into five-year age groups, and combining those 65 years and older at diagnosis in to a single group.

To determine five-year relative survival in Oklahoma, we first calculated observed survival, then used SEER expected survival life tables[20] from 1997-2008 to calculate relative survival using the methods developed by Dickman 2004 in SAS v. 9.4.[21] In order to have more complete follow-up through November 1, 2014, we limited our years of diagnosis to 1997-2008. We calculated survival by race, age, year of diagnosis (1997-2001 and 2002-2008), and stage. We classified race as white, African American, and other (American Indian/Alaska Native and Asian/Pacific Islander) due to the availability of race

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classifications in the expected survival life tables. Hispanic ethnicity was not available in the expected survival life tables. To compare survival over time, we dichotomized the year of diagnosis into 1997-2001 and 2002-2008 for stability and confidentiality reasons. For age-specific survival, we classified age as 0-14 years, five-year age groups through 59 years of age, and 60 and older, again for confidentiality and stability reasons. We used the same staging classifications for the survival analysis as defined for the incidence calculations. We excluded those cases who were identified by death certificate and autopsy only from the analysis.

We used SEER*Stat software to obtain incidence rates and five-year relative survival for SEER data. We used SAS v. 9.4 for all analyses of Oklahoma data. This study was approved by the Institutional Review Boards of the University of Oklahoma Health Sciences Center and the Oklahoma State Department of Health.

RESULTS

Incidence

The AAIR for testicular cancer in Oklahoma was 4.9 per 100,000 among those diagnosed between 1997 and 2012, which was lower than the AAIR in the US (5.5 per 100,000) (Table 1). In Oklahoma, 65.3% (n=842) were diagnosed at local stage, 17.2% (n=222), were diagnosed at regional stage, and 11.7% (n=151) were diagnosed at distant stage (5.5%, n=71 unknown) from 1998-2012. This distribution was similar to that in SEER data during this period (Local: 66.7%, n=21,923; Regional: 17.2%, n=5632; Distant: 11.2%, n=3,671; Unknown: 4.7%, n=1,547). In the US and Oklahoma, the incidence was highest in white NH (US: 7.4 per 100,000; OK: 5.4 per 100,000), followed by American Indians/Alaska Natives (AI/AN) NH (US: 5.6 per 100,000; OK: 5.3 per 100,000), and Hispanics (US: 4.3 per 100,000; OK: 4.1 per 100,000). The AAIRs by race/ethnicity were similar in the US and Oklahoma with the exception of white NH, where the AAIR was lower in Oklahoma (5.4 per 100,000) than in the US (7.4 per 100,000). AAIR were also similar by year of diagnosis, however, the AAIR in Oklahoma was lower for those diagnosed from 2007-2012 (5.1 per 100,000) compared to the US (5.8 per 100,000). In both Oklahoma and the US, incidence was highest among those between the ages of 25 and 40 years of age. Oklahoma men diagnosed at ages 15-19 years (2.5 v. 3.8 per 100,000), 20-24 years (7.7 v. 9.9 per 100,000), and 25-29 years (11.4 v. 13.5 per 100,000) had a lower age-specific incidence rate than the US, but no other age-specific differences were present.

Survival

Overall, the five-year relative survival from testicular cancer was 95.3% in the US and 93.6% in Oklahoma (Table 2). Based on the overlapping 95% CI, we observed no differences between the US and Oklahoma regarding survival by year of diagnosis, race, age, and stage. There was no difference in the US for survival by time period. Regarding race, those with unknown race had the highest relative survival in the US (99.1%), followed by white (95.6%), other (91.4%), and African Americans (88.9%). In Oklahoma, those of unknown race (100.0%) and African Americans (98.5%) had the highest relative survival, although the confidence intervals overlapped the estimates for other racial groups. In both

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the US and Oklahoma, survival was lowest among those 60 years and older (US: 84.4%, OK: 80.5%). Survival was above 70% regardless of stage, but highest among those diagnosed at local stage (US: 97.4%, OK: 97.4%).

DISCUSSION

Testicular cancer is rare in Oklahoma and the US and survival is above 90%. Age-adjusted incidence rates of testicular cancer were higher in the US than in Oklahoma, particularly among white NHs, men 15-19 years of age, and men 20-24 years of age. However, survival did not differ by year of diagnosis, race, age at diagnosis, and stage in Oklahoma compared to the US.

Caucasian men get testicular cancer at a rate five to six times higher than African American men.[9] The reasons for this are largely unknown, although recent research has demonstrated an association between a single nucleotide polymorphism affecting a p53 binding site on the KITLG gene and the development of testicular cancer.[22] This single-nucleotide polymorphism (SNP) is observed at a much higher rate in European Caucasians than in those of African descent. This SNP may have been naturally selected for in European Caucasians, as it also plays a role in protection from UV damage in light-skinned individuals.[22]

While Caucasian men are more likely to develop testicular cancer, studies have demonstrated that non-Caucasian men tend to have worse outcomes.[23,24] A review of 215 patients with testicular cancer found no differences in tumor type nor stage at presentation between white and non-white men. This same analysis showed that there was no statistically significant difference in survival between Hispanic and white patients, but that African American patients had a 17% decrease in survival compared to white patients.[24] Furthermore, another study reported that African American race and low SES appeared to predispose men to more advanced disease stages, and that men of African American race and men with low SES had higher overall mortality and cancer-specific mortality than Caucasian patients.[23] More recent research has shown that testicular cancer-specific survival between races is converging over time, and suggests that diagnostic and treatment discrepancies between racial groups may be improving for non-Caucasian patients.[25]

An advantage of this study was the ability to obtain data from population-based registries in Oklahoma and the US to evaluate a rare cancer. Both registries are high quality and have data available over a long period of time, particularly SEER. Furthermore, we were able to use the same data sources to compare both incidence and survival. One limitation to our survival analysis was the inability to calculate relative survival for AI/AN, Asian/Pacific Islanders, or the Hispanic population due to limitations in the expected survival tables. This prevented us from understanding how survival differs by all race and ethnic groups, particularly AI/ANs, which account for 9.0% of the population in Oklahoma compared to 1.2% in the US.[26] Furthermore, while the OCCR conducts follow-up on all cases of cancer included in the registry, some cases may be lost to follow-up due to migration out of the US. Because the OCCR links with the Oklahoma Mortality Data, the Social Security Death Index, and the National Death Index, vital status is more complete accounting for lag time

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for death reporting. However, because OCCR does not link with the National Death Index each year, the survival may be overinflated. For this study we used the presumed alive methodology as it has been shown to be comparable to SEER follow-up methods.[27, 28]

Testicular cancer is a rare cause of cancer, both in terms of incidence and in cancer deaths. Marked disparities exist in the development of testicular cancer, with Caucasian men being at the highest risk. There is little that can currently be done in terms of prevention since etiology is unknown, and the current guidelines recommend against screening for this disease. Fortunately, most cases of testicular cancer are diagnosed at an early stage, and prognosis is excellent in terms of five-year relative survival, even in those with late-stage disease. Future studies should consider evaluating incidence among multiple states in a region to better understand time trends at a smaller geographic level than the entire US.

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Table 1

Testicular cancer incidence rates per 100,000 for Oklahoma and the US by race/ethnicity, year of diagnosis, and age

Characteristic	SEER Incidence Rate per 100,000 (95% CI)	Oklahoma Incidence Rate per 1000,000 (95% CI)
Overall[a]	5.5 (5.5, 5.6)	4.9 (4.7, 5.2)
Year of Diagnosis[a]		
1997-2001	5.3 (5.1, 5.4)	4.7 (4.2, 5.2)
2002-2006	5.5 (5.3, 5.6)	5.1 (4.7, 5.6)
2007-2012	5.8 (5.7, 5.9)	5.1 (4.6, 5.5)
Race/Ethnicity[a]		
White non-Hispanic (NH)	7.4 (7.3, 7.5)	5.4 (5.1, 5.7)
African American NH	1.5 (1.4, 1.6)	1.4 (0.9, 1.9)
American Indian NH	5.6 (4.8, 6.4)	5.3 (4.4, 6.2)
Asian/Pacific Islander NH	2.2 (2.0, 2.3)	1.9 (0.1, 3.6)
Hispanic	4.3 (4.1, 4.4)	4.1 (3.0, 5.1)
Age		
0-14 years	0.3 (0.2, 0.3)	0.2 (0.1, 0.3)
15-19 years	3.8 (3.5, 4.0)	2.5 (1.9, 3.2)
20-24 years	9.9 (9.5, 10.3)	7.7 (6.5, 8.8)
25-29 years	13.5 (13.0, 13.9)	11.4 (9.9, 12.9)
30-34 years	13.5 (13.1, 14.0)	13.6 (11.9, 15.3)
35-39 years	11.5 (11.1, 11.9)	10.9 (9.4, 12.3)
40-44 years	9.2 (8.8, 9.6)	8.4 (7.1, 9.7)
45-49 years	6.4 (6.1, 6.8)	5.5 (4.5, 6.6)
50-54 years	4.1 (3.8, 4.4)	4.1 (3.2, 5.0)
55-59 years	2.5 (2.3, 2.8)	2.1 (1.4, 2.8)
60-64 years	1.8 (1.5, 2.0)	2.1 (1.3, 2.9)
65+	1.1 (1.0, 1.2)	1.1 (0.7, 1.4)

[a]Rates age-adjusted to the 2000 US Standard Population

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Table 2

5-year relative survival estimates for testicular cancer for Oklahoma and the US by race/ethnicity, year of diagnosis, age, and stage

Characteristic	SEER 5-Year Relative Survival (95% CI)	Oklahoma 5-Year Relative Survival (95% CI)
Overall	95.3 (94.9, 95.6)	93.6 (91.9, 95.3)
Year of Diagnosis		
1997-2001	95.4 (94.8, 95.9)	93.1 (90.3, 95.9)
2002-2008	95.2 (94.7, 95.6)	93.9 (91.7, 96.0)
Race		
White	95.6 (95.2, 95.9)	93.8 (92.0, 95.6)
African American	88.9 (86.0, 91.3)	98.5 (89.5, 100.0)
Other	91.4 (89.2, 93.1)	90.1 (84.2, 96.0)
Unknown	99.1 (96.6, 99.8)	100.0 (100.0, 100.0)
Age		
0-14 years	96.7 (93.1, 98.4)	100.0 (100.0, 100.0)
15-19 years	92.6 (90.7, 94.0)	97.9 (93.0, 100.0)
20-24 years	94.1 (93.1, 95.0)	96.6 (93.2, 100.0)
25-29 years	95.8 (95.0, 96.4)	94.5 (90.8, 98.2)
30-34 years	96.0 (95.3, 96.7)	91.7 (87.7, 95.8)
35-39 years	96.3 (95.5, 96.9)	95.2 (91.5, 98.9)
40-44 years	96.0 (95.1, 96.8)	92.2 (87.1, 97.3)
45-49 years	95.3 (94.0, 96.3)	94.9 (89.1, 100.0)
50-54 years	95.8 (94.0, 97.1)	93.6 (85.1, 100.0)
55-59 years	92.9 (89.5, 95.3)	94.5 (80.7, 100.0)
60+ years	84.4 (79.3, 88.4)	80.5 (63.9, 97.1)
Stagea		
Local	99.2 (98.9, 99.4)	97.4 (95.8, 99.0)
Regional	95.7 (94.9, 96.4)	92.2 (87.5, 96.9)
Distant	72.0 (70.1, 73.8)	75.9 (67.2, 84.7)
Unknown	85.9 (81.1, 89.5)	85.7 (75.4, 95.9)

[a]Survival by stage is limited to 1998-2008 to be consistent with the SEER Summary Stage 2000 variable.